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DISEASES OF THE NERVOUS SYSTEM

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Psychotic Reactions Following Sulfanilamide Administration*

FRANCIS J. TARTAGLINO, M.D.
Washington, D. C.

Mental disorders following the administration of sulfanilamide were first brought to our attention at Saint Elizabeth's Hospital about October, 1939. A colored patient aged 21, diagnosed dementia praecox, catatonic type, in a condition in which all the psychotic symptoms had cleared up, received sulfanilamide therapy for an acute gonorrheal urethritis. After ten days of treatment during which the patient had received a total of 480 grains of sulfanilamide, the patient again developed mental symptoms. His former delusional ideas not only returned, but he gave expression to many others, symptoms not prominently present previously. This patient had not refused food during his previous psychotic episode, but it became necessary at this time to tube-feed him. As soon as the drug was discontinued, the patient promptly recovered. It was our opinion that sulfanilamide was probably the cause of the acute exacerbation of the mental symptoms. Patient himself thought that the medicine he took for his acute gonorrheal urethritis was the cause of his excitement.

Shortly after this it was necessary again to subject a dementia praecox patient in remission to sulfanilamide therapy for a gonorrheal infection, and again, following the administration this time of 460 grains of sulfanilamide over a 10-day period, mental symptoms were observed.

One may be justified in holding the opinion that it was a mere coincidence for both of these patients in remission to have relapsed to their previous psychotic condition. However, within the next six months we observed other cases in which a causal relationship between the intake of sulfanilamide and their psychosis appeared to be certain. We find it, therefore, useful considering the very extensive use of sulfanilamide to record all of our observations.

We divide our cases into two groups: Group No. 1—patients in whom we feel that the untoward effects of the drug were the direct cause of their admission to a mental hospital. Group No. 2—patients who, during remissions of their psychoses, again developed mental symptoms following administration of the drug.

Group No. 1—The first case is that of a colored boy aged 17, who was picked up by the police on March 5, 1940, in a pool room, with the report that he had suddenly become violent and hallucinated. In the Municipal Hospital, where he was first taken, he was described as being extremely confused, disoriented, and markedly disturbed. He expressed some vague delusional ideas of his father attempting to kill him. When the patient was admitted to Saint Elizabeth's Hospital on March 12, 1940, he was still a little confused. He recalled, however, being violent at the Municipal Hospital. He became violent because he was afraid; kept hearing people outside of his room saying they were going to get a gun and kill him. The patient's memory was still poor and he was still disoriented,

*From St. Elizabeth's Hospital, Washington, D. C.

being unable to give the day, the month or the year. Apprehension was marked, and there was some intellectual impairment. A history was obtained as follows:

About the middle of February the patient had acquired a gonorrheal infection, and he had been given some white pills to take at one of the venereal clinics in town. "Taking too many of these pills made me wild," he said. A report from the venereal clinic showed that the patient had received a total of 750 grains of sulfanilamide over a 19-day period. He was still taking this medication on the day he was picked up by the police. On March 16, 1940, four days after admission to St. Elizabeth's Hospital and eleven days after his last dose of sulfanilamide, we found this patient improved mentally. He was less confused, less apprehensive and he became oriented gradually. He was described as being generally more cooperative when it was again necessary to start him on sulfanilamide therapy, because of a recurrent purulent discharge. After the patient had received 180 grains of sulfanilamide he developed a spiking type of temperature reaction averaging 103/6 on three successive days. Sulfanilamide therapy was continued for six days more, during which time he had received a total of 570 grains; because of his extreme confusion and excitement again, the drug was withdrawn. A nurse's note on this occasion is interesting: "The condition of this patient has changed very much during the past week. Last week a very cooperative patient, now he is very confused. He insists on going about nude and is being tube-fed. He refuses all medication and all day long he paces back and forth in his room, talking and singing, and he is very noisy." The physician on the case remarked, "This agitated, catatonic-like condition continued for 12 days and then the patient cleared up mentally." This patient, therefore, showed a mental reaction on two different occasions, following sulfanilamide administration, the psychosis each time lasting about two weeks.

The second case is that of a 21-year-old colored patient, a former Mess Attendant, U. S. Navy, whose mental illness began about January 13, 1940, when he was observed as being depressed and unusually seclusive while on active duty. When questioned by the Navy physician he expressed numerous somatic complaints referable to his gastrointestinal tract and felt he was going to die almost any minute. He was unable to carry on a connected conversation, and there were both auditory and olfactory hallucinations. In addition, he showed some suspiciousness, ideas of reference, thought blocking, retardation, and disorientation. On admission to Saint Elizabeth's Hospital on February 22, 1940, he told the following story:

He reported to sick bay on one of the United States Naval vessels, suffering from an acute gonorrheal infection, December 31, 1939, and was examined and treated with sulfanilamide. Shortly after taking the medication he developed a pain in his back and began to feel "foolish." He became afraid of everything, felt jumpy and shaky. He was placed in sick bay for four or five days, during which time, he states, he was very confused and did not know what was going on. He jumped overboard on or about January 22, 1940, because it seemed to him at the time that all the gunners were training their guns on him and were going to kill him. He was swimming toward shore when a boat was lowered and after some struggle he was pulled out. He stated, "I was mean and wanted to fight everybody—I was dizzy, confused, and I heard voices of both men and women."

Information from the Medical Officer of the Naval vessel on which the patient was treated states that the patient had been given a total of 780 grains of sulfanilamide over a 16-day period—from December 31, 1939, to January 15, 1940—when the drug was discontinued. The mental symptoms were first noted on January 13, 1940. This patient remained mentally ill approximately five weeks following the discontinuance of the drug. It was noted when he was examined, soon after admission to Saint Elizabeth's Hospital, that he was well on the road to recovery.

The third case is that of a young white male aged 18, who, on complaint of relatives, was admitted to a Municipal Hospital for mental observation on February 4, 1940. He was described at that institution as showing the following symptoms: Seclusiveness, preoccupation, listlessness, mannerisms, rigidity, resistiveness and uncooperativeness. He was completely out of touch, very confused and wandered aimlessly about the ward; in addition, it is said that at times he assumed awkward positions, and exhibited impulsive and stereo-

typed conduct. He was frequently mute, often was found weeping and then smiling, and was so noisy and excitable at times as to require restraint. Four days later, on February 8, 1940, patient was admitted to Saint Elizabeth's Hospital in a highly excited condition, and when seen by the admitting physician soon afterwards, was found nude in his room. He had overturned furniture and had torn his bed apart. He was confused and irrelevant. There was evident thought blocking and disorientation in all fields. Patient complained of a buzzing and a roaring sensation in his ears. From relatives we obtained the following information:

About February 1, the patient was found by his sister crying and very much upset. He complained that his head hurt and felt as if it were on fire. He was ordered to bed by the family physician and soon afterwards he was noted as talking foolishly and apparently much confused. He began making queer noises with his lips, snapped his fingers continually, got up out of bed, urinated in his clothing and walked about in a puddle of urine on the floor. He was promptly put to bed again and he did not seem to have the use of his hands. He was up again and urinated on the floor. He did not recognize his relatives when they came to see him. The relatives reported that for about three weeks prior to the patient's mental illness he had been under treatment for a gonorrheal infection with sulfanilamide. The infection was acquired on January 1, 1940, and the patient went to a venereal clinic where he was started on sulfanilamide therapy on January 9. He was told to take 15 grains three times a day and he continued this until the onset of his illness about January 31 when, as he has since expressed it, "I blew up, couldn't sleep. My brain got a fever. I felt like my brain was burning up and I kept pacing up and down the floor." Patient stated that he had an amnesia for events at the Municipal Hospital, and he did not recall coming to the hospital or being brought in strapped to a stretcher. Over a 17-day period he had received a total of 765 grains of sulfanilamide. He was still ill mentally upon admission. A purulent discharge was present and, unfortunately, he was again started on sulfanilamide therapy. An adequate history had not as yet been obtained by the hospital, hence it was not recognized that this drug was possibly the cause of the patient's disturbance. Over a 16-day period at this institution he received a total of 980 grains of sulfanilamide and the patient was seriously disturbed throughout this time. He was not only troublesome, but the symptoms previously described were now even more in evidence. Two weeks after sulfanilamide was discontinued the patient was found to be improving and progressing mentally. He described an amnesia for his disturbed period. This patient's psychosis lasted approximately six weeks. There is no doubt, however, that it would have been much shorter had he not again been given sulfanilamide.

The fourth case is that of a 24-year-old white sailor. He reported ill on active duty November 18, 1939, and was found to have a gonorrheal infection. He was promptly placed on sulfanilamide therapy. On December 5, 1939, he again reported ill, this time with gastrointestinal complaints. He was having pain in his abdomen associated with slight nausea and general malaise, and the diagnosis was acute gastroenteritis. On December 10, 1939, 23 days later, he was placed under mental observation with the report: "This patient is found having frequent crying spells. He is unable to carry on his duties, unable to concentrate or carry on an intelligent conversation." Up to this time patient had received 517½ grains of a sulfanilamide product called P-Sulphanil-Acetyl-Imide (Trade No. 338-A), and, of his own volition, he confessed, he had taken about 300 grains of sulfanilamide the previous month. On December 15, he was again started on a course of sulfanilamide, over a 7-day period, and received 315 grains more of the drug. On January 3, 1940, this patient was exhibiting many ideas of reference and was having both auditory and visual hallucinations. He also described feelings of something crawling in his rectum and pain in his perineum. He was disoriented, confused and clouded mentally. The patient was ill mentally from this time on, showing self-accusatory ideas, a disordered attention, making several attempts to injure himself, and was combative. Then on February 10, it was noted that he was quiet, although showing limited interest but obviously still somewhat depressed. On February 22, he was again given prontylin therapy. He continued this up until about March 11, receiving a total of 810 grains

of the drug over a 17-day period. Patient was then admitted to Saint Elizabeth's Hospital March 18. When questioned on admission he described his mind as being "in a fog" and feeling as if he were "in a coma." The physician noted that he talked and walked slowly, showed some spontaneous laughter, and at times was dull and retarded. As the patient began to improve he described a period of amnesia for many events while taking the drug. He stated "Things began to fade away. The sensation was just like gradually getting drunk—I didn't realize what I was doing when I began banging my head. I thought I was going to die and I got the wrong impression of everything. I had a vivid imagination—imagined a lot of crazy things and I was afraid." He made the interesting comment, "They should have kept me at the Naval Hospital and returned me to duty after I got over the bad effects of the drug." While in the service, the patient had been given about 1425 grains of sulfanilamide and 517½ grains of P-Sulfanil-Acetyl-Imide over a period of about 46 days, in addition to the 300 grains the patient had taken voluntarily. It is noted that he was given sulfanilamide on three separate occasions, each time reacting with acute mental symptoms. It is difficult to say just how long the psychosis lasted in this case, the picture being complicated by the fact that he was repeatedly given sulfanilamide.

Group No. 2—For the sake of brevity, cases in Group No. 2 will not be described in detail. These are the cases that first came to our attention; all were cases of catatonic dementia praecox who were treated with sulfanilamide while in a remission and reacted unfavorably to the drug. There are three cases in this group.

The first was given a total of 480 grains over a 10-day period; a second 460 grains over a 10-day period, and a third 395 grains over an 8-day period. The symptoms presented generally by the patients were the same as already described in cases under Group No. 1. In addition they had such signs of toxicity as gastrointestinal complaints, dizziness, headache and a period of amnesia during the height of their disturbed state. Each of these patients promptly showed improvement mentally in from two to three weeks following discontinuance of the drug.

Review of the Literature

Many and varied toxic complications resulting from the use of sulfanilamide have been reported, but there have been few reports bringing forward the fact that the drug is responsible for mental disorders. Lieutenants Hogan and McNamara[1] of the Medical Corps of the U. S. Navy, should be credited with making the first report of a case. It was the case of a Private, U. S. Marine Corps, aged 21, who, while under treatment for an acute gonorrheal urethritis, received between August 30, 1937, and September 24, 1937, a total of 1,220 grains of sulfanilamide, following which a psychotic picture developed, characterized at first by a mild paranoid "feeling," with apprehension and restlessness, and later on by both auditory and visual hallucinations. The picture, they said, resembled a toxic delirium with dream-like fantasies and bewilderment. Pearson and Burnstine[2] of the Philadelphia General Hospital have reported two cases; the first of a man aged 29 who, while receiving treatment for acute gonorrheal urethritis, took a total of only 270 grains of sulfanilamide over a 3-day period and developed such signs as confusion, disorientation, retardation, negativism, bizarre behavior with mild paranoid trends. The second case is that of a young colored girl aged 18 who received a total of 710 grains over an 8-day period, and had such mental signs as clouded sensorium, bewilderment, rapid mood changes, distractibility, hallucinations of a certain nature and delusions. Both of these patients showed premonitory signs such as headache, dizziness and lethargy. Rapid recovery followed withdrawal of the drug in both cases.

A very interesting report is presented by Young[3] in the *Ohio State Medical Journal*, August, 1939, of a 37-year-old white male who was found by the police amnesic and wandering about. There was complete loss of memory for past events, the patient did not know his name, address, relatives or the date. Neurologically, he showed fixed pupils, positive Romberg, irregular tendon reflexes and a bilaterally suggestive Babinski sign. This condition remained unchanged after 72 hours. A history was obtained from the family of progressive mental deterioration over a period of three weeks' time. After 96 hours the patient was mentally clear and a neurological

examination was normal. He then gave a history of voluntarily taking sulfanilamide. He was not clear on the dosage but treatment started three weeks prior to the onset of amnesia and it is probable, the author states, that the dose was from 90 to 150 grains daily, at least for the first week.

W. H. Block[4] reported a case of a white male aged 63, previously mentally sound, who, while under treatment for an acute respiratory infection, streptococcic in origin, was given neoprontosil, grains 20, every four to six hours. By the time the patient had taken four doses a state of mental confusion was noted, which was at the time attributed to the infection. Neoprontosil was continued for three more doses, making approximately 135 grains taken in all. The patient then became uncooperative, would not stay in bed, stating that he had been up the day before and complained bitterly of the rain outside, even though the day was clear. He complained of wires hanging from the ceiling and birds in a tree outside his window, which did not exist. He also thought that he was in the home of one of his daughters instead of being in his own home. Neoprontosil was discontinued and all mental symptoms cleared within 12 hours. The patient had an amnesia for the events during this period. Dr. Block, in his summary, comments: "The rapid clearing of the mental symptoms following cessation of the drug proves without a doubt that neoprontosil was the precipitating factor."

Townsend and Mulcahy[5] of New York City report on a study of 91 men treated with sulfanilamide for acute gonorrheal urethritis, and all were found to have shown some toxicity. They recorded six patients of their group as being markedly toxic, 24 as being moderately toxic, and 64 slightly toxic. They cite a long list of symptoms but only those referable to the central nervous system are given here; they include headache, nine cases; vertigo, 81 cases; insomnia, 16 cases; drowsiness with slight disorientation, 13 cases; paresthesias, 5 cases; tinnitus, 16 cases, and diplopia, 3 cases.

One group of authors in writing on the toxicity of the drug states, "While sulfanilamide has relatively low toxicity for animals,[6] certain individuals respond in a way that is exceedingly alarming and indeed dangerous." Another author[7] in discussing the use of sulfanilamide in puerperal fever, states "A number of symptoms referable to the nervous system were occasionally observed, including depression, headache, dizziness, blurred vision and spots before the eyes, and paresthesia of the peripheral nerves, as well as mental disturbances of a hysterical type."

We are aware that most patients using sulfanilamide may at some time or another show a more or less toxic reaction to the drug. Whether or not it causes any permanent damage to the central nervous system is a question that has not as yet been definitely settled. There are reports on two cases of encephalomyelitis following administration of sulfanilamide, one of which ended fatally.[8] In both cases, however, the author thinks the condition was due to idiosyncrasies rather than to overdosage. Post-mortem findings in the first case were perivascular demyelination in the thoracic cord, vascular damage and massive softening of the lumbar cord. The author states that in both cases lesion of the central nervous system developed during treatment with sulfanilamide. In Case No. 1, the symptoms suggested an acute ascending myelitis; in Case No. 2 the clinical findings were consistent with encephalomyelitis.

In an experimental study of the pharmacological properties of sulfanilamide made on animals, the central nervous system was examined on three of those that died from the drug.[9] The animals used were cats, dogs and rabbits. There were degenerative changes; that is, chromatolysis in the neurons of the anterior column of the spinal cord and in some of the nerve cells of the cortex and midbrain. Experimentors state that the symptoms suggested that in acute poisoning by large doses of sulfanilamide the stress falls mainly on the central nervous system, apart from the dyspnea. In cats there appeared to be a transient paralysis of the cerebral cortex for a few hours; in rabbits the cortex was only depressed. The authors state extensor rigidity of the limbs and nystagmoid movements of the head are evidences of disturbed functions in the brain-stem and spinal cord, but how far these are due to excitation and how far to removal of inhibitions is difficult to decide.

Discussion

The symptoms found in our cases were in most instances consistent with the findings of those described by the authors mentioned.

All of our cases showed toxic signs consisting of G. I. complaints of nausea, vomiting, anorexia, in addition to dizziness, headache, drowsiness and some fever prior to the psychotic symptoms. Of the five cases described in a review of the literature, four showed sensorial defects, all showed confusion and general hyperactivity, three had hallucinations—either auditory or visual—four, delusions of persecution and ideas of reference, three excitement and three an amnesic period. There were also some scattered symptoms such as negativism, suspiciousness, dullness and apathy, bizarre behavior and mood changes. All of the above symptoms were not only found in our cases but as well there were such pertinent findings as fear and apprehension, which were present in all of our cases, depression in three cases, suicidal ideas in three cases, a strong fear of impending death in four cases, impulsive tendencies and an amnesic period in all cases.

One may wonder why these few patients developed mental disorders while so many have escaped. It is a difficult question to answer; it may be that the constitutional make-up and personality of the patients who did develop mental disorder were such that they would have developed a mental disorder under some other stress or strain of everyday life. Certainly, the doses of sulfanilamide given our patients previous to and following admission to our hospital were by no means extraordinarily large. On the other hand, it is to be noted, particularly in our cases in Group No. 1, that had therapy been discontinued in ten days' time, probably no psychosis would have resulted. In passing, we question whether these patients should have received treatment with sulfanilamide over a longer duration than ten days, when it was obvious that sulfanilamide was apparently a failure in bringing about a cure of their gonorrheal infection. I mention this because many urologists have agreed that if there are no beneficial results within ten days' time the drug should be discontinued.[5,10]

Two of our cases definitely, and there is a suggestion of a third, drank alcohol while under treatment with sulfanilamide. The amount of alcohol taken was not excessive, yet it probably tended to accentuate the toxic cerebral symptoms. This is in agreement with statements made by others.[11] We feel, therefore, that alcohol is contraindicated during sulfanilamide administration.

If one realizes the toxic cerebral symptoms to be expected from the use of this drug and what serious consequences may result from having these symptoms superimposed on an already existing mental illness, we believe in view of our present findings and observations, that it would be most wise for those who use sulfanilamide to administer the drug with extreme caution, especially to any person possessing an unstable temperament or in any suspected case of organic brain disease such as senile psychosis, arteriosclerosis, alcoholism, paresis, etc.

Summary and Conclusion

1) A total of seven cases are presented, four cases in Group No. 1, in which it is felt that admission to a mental hospital is directly due to sulfanilamide administration. Three cases are presented of an already existing psychosis and who, during a remission, were given sulfanilamide which produced further mental symptoms. Sulfanilamide, therefore, in these cases interfered with convalescence.

2) Characteristic symptoms were those of delirium, delusions—both visual and auditory—a strong fear of impending death, sensorial defects, marked confusion and excitement, and subsequent amnesia for a period of time.

3) We do not mean to imply here that sulfanilamide should not be continued in any patient showing marked toxicity to the drug. What we desire to convey is when a patient shows toxic symptoms it indicates that he does not tolerate the drug well, and if sulfanilamide is to be continued, it should be done cautiously and under close supervision by the physician. If decreasing the dose does not alleviate mental symptoms, it would be best to institute a therapy other than sulfanilamide. On the other hand, there is enough evidence to show that the drug, to prove effective, must be given in high amounts. The case reports just described should not prompt the physician to become too conservative in the future administration of the drug. One should remember that in any new form of therapy untoward effects must always be expected. These, however, can be kept to a minimum if the patient is treated judiciously and with consideration.

References

1 Hogan, B. W., and McNamara, P. J.: "Psychosis Precipitated by Sulfanilamide," from the *U. S. Naval Medical Bulletin*, 36: 80, (Jan.) 1938.

2 Pearson, Manuel M., and Burnstine, David M.: "Psychosis Precipitated by Sulfanilamide, Report of Two Cases," from *International Clinics*, 3: 246, (Sept.) 1939.

3 Young, Robert E. S.: "Psychosis Due to Sulfanilamide," from *Ohio State Medical Journal*, 35: 847, (Aug.) 1939.

4 Bloch, W. H.: "Psychosis Precipitated by Sulfanilamide," *Journal of the Medical Association of Alabama*, 9: 339, 1939-1940.

5 Townsend, Terry M., and Mulcahy, T. M., New York City: "Role of Sulfanilamide in Acute Gonorrheal Urethritis," from the *New York State Journal of Medicine*, 38: 833-835, (June 1) 1938.

6 Eggleston, Cary, and Weiss, Soma: "Toxicity of Sulfanilamide," from the *American Journal of Medical Science*, 195: 701-706, (May) 1938.

7 Colebrook, L., and Purdie, Anthony, Queen Charlotte Hospital: "Sulfanilamide in Puerperal Fever," from the *Lancet*, 2: 1237, (Nov. 27) 1937; 2: 1291, (Dec. 4) 1937.

8 Fisher, J. H., and Gilmour, J. R.: "Encephalomyelitis Following Administration of Sulfanilamide, With a Note on Histological Findings," from the *Lancet*, 237: 501, (Aug. 5) 1939.

9 Hawkins, F.: "A Study of the Pharmacological Action and Properties of Sulfanilamide," pp. 38-39 of 1938 *Year Book of Therapeutics*.

10 Mathe, Chas. P., and Spitalmy, August: p. 67, 1938 *Year Book of General Therapeutics*.

11 Long, Perrin; Bliss, Eleanor, and Feinstone, W. Harry, Johns Hopkins University: "Mode of Action, Clinical Use and Toxic Manifestations of Sulfanilamide," from the *Journal of the American Medical Association*, 112: 115-121, (Jan. 14) 1939.

12 Pelouze, P. S.: "Oral and Medical Treatment of Gonorrhea in the Male," pp. 1878-1879 of the *Journal of the Medical Association*, Vol. 114, No. 19, (May 11) 1940.

13 Hering, E. R., Lieut., Medical Corps, U. S. Navy: "Treatment of Gonorrhea with Sulfanilamide," from the *United States Medical Bulletin*, p. 63, Jan., 1938.

14 *Urology and Cutaneous Review*, 42: 79-81, (Feb. 1) 1938.

15 Farrell, Lyman and Youman, Northwestern University: "Sulfanilamide in Gonorrheal Urethritis," from the *Journal of the American Medical Association*, 110: 1176-1177, (April 9) 1938.

Surgical Treatment of Epilepsy*

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Craniotomy has a longer history than any other surgical procedure. The history of epilepsy is as old as that of man himself. It is not surprising, therefore, that the early history of craniotomy and epilepsy are inextricably interwoven. Evidences of trepanation have been found in prehistoric skulls and there is every reason to assume that these trepanations were designed to provide an exit for evil spirits. These evil spirits were probably the accepted cause of insanity and epilepsy. Today we are still carrying out operative procedures on cases of insanity and epilepsy, with however a quite different rationalization.

Trepanation is still practiced among certain primitive people. Brodsky[1] recently reported on the trephiners of Blanche Bay in New Britain and gave an interesting description of their instruments and methods. Probably these are not essentially different from those used by prehistoric trephiners. Brodsky stated that these native "surgeons" have a mortality rate of about 30 per cent which he compares favorably with that of 75 per cent mortality reported by Hudson for 32 trephanations in St. George and Guys Hospitals during the period 1870-77.

It is interesting to note that Benjamin S. Dudley,[2] Professor of Surgery at Transylvania University, Lexington, Kentucky, operated on five cases of traumatic epilepsy between 1818 and 1827 with no fatalities and four apparent cures. This surgeon was apparently the first in the United States to operate on cases of traumatic epilepsy. Daniel Brainard[3] of Chicago in 1858 reported five cases trephined for epilepsy or insanity with no deaths, and cure or improvement in four cases. When Billings[4] in 1861 collected 72 cases from the literature, Dudley and Brainard were the only surgeons credited with more than three cases. With these two outstanding exceptions, mortality was of course high because of the frequency of infection and consequent meningitis.

Modern neurosurgery waited the development of asepsis, cerebral localization, and anesthesia. Victor Horsley's first craniotomy in 1886 was for the relief of post-traumatic Jacksonian epilepsy and a cure resulted. Since that time many craniotomies have been car-

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ried out on patients with seizures but no surgeons have reported larger series of cases than Penfield.[5] This author's reports and finally his book on "Epilepsy and Cerebral Localization" furnished the impetus to many other surgeons to continue work in this field.

With increasing knowledge regarding seizures it has become possible to divide and classify them into groups and subgroups depending on their clinical type, the anatomical localization of the lesion, and the etiology of the causative lesion. Recently electroencephalographic studies have led to attempts by various authors such as Gibbs and Jasper[6] and Kershman[7] to classify seizures on the basis of the electroencephalogram. However, it has not been possible so far to accurately correlate the various types of abnormal patterns seen in the electroencephalograms of epileptic patients with corresponding types of clinical seizures.

Seizures that are likely to be benefited by surgery fall into two groups: those due to an expanding cerebral lesion and those due to some localized atrophic epileptogenic lesion. The three common types of expanding lesions are neoplasm, brain abscess and subdural hematoma. Seizures are common in all of these. It has been long emphasized by neurologists that seizures beginning after 30 years of age are strongly suspected of being of tumor origin.

Parker[8] found seizures in 21.6 per cent of 313 cases of brain tumors at the Mayo Clinic. Groff[9] reported that 30.9 per cent of 291 meningiomas from Cushing's series had seizures. Davis and Droegemuller[10] reported 39 per cent occurrence of seizures in meningiomas and 62 per cent and 72 per cent in glioblastomas and astrocytomas, respectively. The same authors reported that 15 of 42 patients with abscess had fits. Penfield reported an incidence of 37 per cent in 703 cases of tumor. The average incidence reported by other authors ranges from 29 to 39 per cent. All authors agree that seizures are rare in infratentorial tumors, that they are most common in frontal, parietal and temporal tumors and that the nearer tumors are to the central sulcus the higher the incidence of seizures. Slow growing tumors have a greater incidence of seizures than rapidly growing ones. This is probably related to the longer period of time that patients have to develop seizures in.

Brain abscess seems to cause seizures in a higher proportion of cases than tumor. Penfield reported that 27 or 50 per cent of 54 patients with brain abscess had epileptic seizures. Subdural hematoma has a much smaller incidence of seizures. Kunkel and Dandy reported an incidence of 12 per cent in 48 cases, Penfield 25 per cent in 71 cases. Table I gives the number of cases of seizures in the author's experience with verified brain tumors, abscess, and subdural hematoma. The percentages are about the same as reported by other authors.

TABLE I

SEIZURES IN EXPANDING CEREBRAL LESIONS

Lesions	*Verified Cases*	*Cases with Seizures*
Brain tumor	132	26 (19.7%)
Brain abscess	29	13 (44.8%)
Subdural hematoma	69	14 (20.3%)

Localized atrophic epileptogenic lesions have been classified by Penfield as cerebral cicatrix, local cerebral atrophy, local microgyria and brain cyst. This author and his associates have studied thoroughly the effects of various types of trauma on the brain and the different types of scars produced by healing. Injury to the brain in general results in destruction of neurones and overgrowth of neuroglia provided that enough circulation is preserved to provide for reaction and repair. Otherwise the injured portion may disappear leaving a fluid-filled cyst. Injured neurones either die or live in an abnormal state; they do not multiply or hypertrophy. Any injury to cerebral tissue results in decrease in volume because the overgrowth of neurolgia never makes up for the total tissue destruction.

Fine avascular adhesions bridging the subdural space between dura and pia-arachnoid may follow the absorption of subdural blood, meningitis, etc. It is doubtful if such lesions are of importance in producing convulsive seizures. Meningo-cerebral cicatrices may follow injury, drainage of an abscess or a local meningeal infection. These are scars with intimate connections between brain and dura containing blood vessels and connective tissue. These are common epileptogenic lesions and

it is believed, cause seizures in more cases than do cerebral cortical scars that do not involve the meninges. These latter follow localized brain injury in which the pia-arachnoid remains intact and consequently the scar never becomes attached to the dura.

Areas of localized cerebral atrophy are less often the cause of seizures. They may follow vascular occlusion, intra-cerebral hemorrhage, aneurysms and hemangiomas, localized encephalitis, localized trauma, or localized compression (old depressed fracture, etc.).

Local microgyria due to birth injury or congenital vascular anomalies represent a special type of atrophic lesion. Large brain cysts are due to vascular occlusions or to severe injury with destruction of the circulation. Softening and liquefaction necrosis of the brain tissue takes place. There is minimal reaction in the surrounding tissue and finally a fluid-filled cavity with its walls lined with glial tissue results.

The patient with convulsive seizures deserves thorough study. As Penfield has stated, the physician who gives phenobarbital to a patient with an operable brain tumor and the surgeon who performs a craniotomy on a patient with seizures due to hypoglycemia are both open to criticism. The history of the attacks themselves, their onset, the seizure pattern, and post-ictal phenomena should be carefully elicited from both the patient and reliable witnesses. Careful general physical and neurological examinations should be made. Roentgenograms of the skull, electroencephalography and in selected cases pneumo-encephalography should be carried out. If no objective evidence of a cerebral lesion is found, the blood sugar curve should be studied, blood pressure studies made, and basal metabolism and other tests as indicated.

A great variety of operative procedures, intra-cranial and otherwise, have been carried out on epileptics. Most of them such as radical colectomy have been forgotten and better so. Removal of a focus of infection and intoxication is only justified when the existence of such a focus can be proven and its removal can be justified for its own sake. Generally no lasting benefit to the seizures will be obtained. Sub-temporal decompression was recommended by Kocher[11] and has been carried out by numerous surgeons without encouraging results. The tendency to temporary improvement after any types of surgical procedure makes prolonged post-operative observation necessary for any critical evaluation of results.

Sachs[12] described a method of sub-pial resection of the cortex in Jacksonian epilepsy, crediting the method to Horsley. He reported nine cures, one death and one case of recurrence in 11 cases of Jacksonian epilepsy. Furlow[13] later reported 16 more cases with two deaths, entire relief in seven cases, definite improvement in six and no change in one case.

Rowe and Watts[14] reported the excision of cortical scars in 20 patients with complete relief from seizures in three cases, marked improvement in ten and no improvement in seven cases. German[15] reported the removal of 29 cortical scars with one death, complete relief from convulsions in ten cases and good improvement in nine cases. Most of the cases in this series that were relieved had had attacks for less than three years before their operation.

Scarff[16] reported three cases which he had treated by lysis of Pacchionian granulations with complete relief in one and marked improvement in two others. Van Wagenen[17] divided the corpus callosum in ten cases of generalized seizures in the hope of limiting the seizures to one side of the body. His results were not particularly encouraging.

Penfield reported 165 cases of craniotomy for atrophic epileptogenic lesions. There were 115 cases of scar resection with 24 cases free from attacks, 55 improved and 34 little or no better. In 35 cases exploration alone was carried out with one case free from attacks, eight improved and 24 no better. There were seven deaths, three cases could not be followed and in five cases a cortical artery was ligated.

The operative procedure itself has certain important differences from craniotomy for other reasons, such as removal of brain tumors. Generally speaking the most suitable patients for operative intervention are those in whom the seizures are clearly related to a localized cerebral lesion. Both the history of the case and the type of seizure are of great importance in establishing this point. Confirmation of the presence of such a lesion should be made by clinical neurological examination, roentgenography, electroencepha-

lography if feasible, and pneumo-encephalography if indicated.

Cooperation of the patient is essential, both for the operative procedure and in after care. Local anesthesia is most desirable in order that the electrical sensitivity of the cortex remain unaltered. It is the writer's usual practice to use local anesthesia after preliminary sedation with moderate doses of morphine and scopolamine, for the osteoplastic craniotomy and during the period of cortical stimulation and resection. A rectal tube is previously installed and when closure of the wound is begun avertin is administered to the patient, usually a dosage of 90 mg. per kilo of body weight. Another method is to supplement the local anesthesia with intravenous sodium pentothal during the closure. In hypersensitive patients pentothal may be used during the injection of the local anesthetic and for the actual "breaking down" of the bone flap. A trained anesthetist is indispensable even though local anesthesia is used. The same careful attention to the patient's condition with frequent charting of the blood pressure, pulse and respiration is as necessary as in any other brain operation. In addition the anesthetist is invaluable in noting and reporting to the surgeon the responses obtained by electrical stimulation of the cortex.

Electrical exploration of the cortex is carried out as soon as the dura is reflected and the exposed portion of the hemisphere has been examined. Either the faradic or the galvanic current can be used, the latter for mapping out the sensori-motor area, the former for reproducing the patient's seizures. We have used several different stimulators but are now using one made for us by Dr. Hummon of the physiotherapy department of the Cook County Hospital. We have a variable wave frequency but usually use a frequency of 57-60 per second. A bipolar electrode is used and the intensity of stimulus is usually that which will give a slight twitch when applied to the exposed temporal muscle.

After the sensori-motor cortex has been mapped out an attempt may be made to reproduce the initial elements in the patient's seizures, whether aura or initial movement. Production of a major seizure should be avoided if possible. Too strong a stimulus should not be used as a physiologic seizure may thus be produced. If the initial element of the patient's seizure cannot be produced, decision as to excision must be based on other considerations, specifically the presence of gross lesions. The size of such lesions, whether or not they are discrete or diffuse, and their location in relation to the motor cortex are factors of prime importance and each case must be divided on its own merits. Excision may be accomplished by sharp dissection in the case of tough scars but usually is best carried out with a small bore sucker. Adjacent gray matter should be left covered with pia so as to preserve its circulation. Hemostasis of larger vessels is best obtained with silver clips—the electro-cautery should be used sparingly because of the danger of thrombosis of adjacent pial vessels. Generally the ventricle is not opened although it may be so dilated and pulled out to the scar that it is necessarily opened.

After complete hemostasis the dura should be closed. Most cases are drained for 24 hours with a penrose drain beneath the bone flap but when the ventricle is opened it is better to close the wound without drainage. Daily spinal punctures are advisable for a number of days in such cases.

The same careful post-operative observation with frequent charting of pulse and blood pressure is as necessary as in cases of brain tumor removals. Pulse and blood pressure must be charted frequently. Signs of post-operative hemorrhage in the shape of decreasing alertness, slowing pulse, progressive neurologic defect and headache must be watched for. The wound must be re-opened and the blood clot evacuated promptly if such complication occurs. However, in these cases there may be conditions simulating a post-operative clot. If seizures occur in the first few days, paralysis may ensue, due to post-ictal circulatory changes. These paralyses clear up gradually but meanwhile cause much concern. Aside from seizures paralysis may occur on the third or fourth post-operative day because of local cerebral edema. This, too, gradually clears up but while present worries everyone. Fortunately the patient usually remains conscious and fairly cooperative which helps to differentiate the condition from that of post-operative hematoma where stupor gradually deepens.

So-called aseptic meningitis with a pleo-

cytosis of polymorphonuclear lymphocytes in the spinal fluid and persistent fever may occur. Organisms are not found on culture or smear of the spinal fluid and the condition gradually clears up. It is more likely to occur in cases where the ventricle has been opened. Repeated spinal punctures are of value.

Phenobarbital is administered to these patients, beginning immediately after operation, to prevent if possible post-operative seizures. It is my practice to continue it in small doses for at least six months after the operation. Unless patients are cooperative and willing to continue with medication and regulation of their life after operation they are not good subjects for operative treatment. The failure of a patient to cooperate in conservative treatment of his seizures before operation is a contraindication to surgical treatment, not an indication as is sometimes claimed.

Twenty-four patients have been operated on because of convulsive seizures. In none of these cases was there an expanding cerebral lesion. Table II gives the etiologic factor in the cases operated on; Table III the operative procedure. In the eight cases that were explored no procedure was carried out on the brain proper. In these cases a small subtemporal decompression was made. Resection means that either a cortical scar, an area of focal atrophy, microgyria, or brain tissue overlying a cyst was removed. The two cases of calcified lesion were: one a sub-cortical calcified hematoma of the left parietal lobe that was removed, the other a sub-cortical calcification of unknown etiology in the right temporal lobe that was likewise removed.

Table III gives the results obtained in relation, respectively, to the age of the patient, the duration of seizures before operation, and the operative procedure used. There was one operative death, due to meningitis. Two cases have been operated on so recently that they were not included in the results and one case has not been heard from since his dismissal from the hospital. The remaining twenty cases have been followed for from six to eight years. Two of these have had no seizures since operation which was in one case two years ago. And in the other six there has been a rare seizure, often only in the immediate post-operative period. Seven cases are improved. In this group the seizures are now well controlled by dilantin or phenobarbital or have been greatly lessened in frequency or

TABLE II

CASES OPERATED ON FOR SEIZURES

Etiology	*No. of Cases*
Trauma	17
Birth injury	3
Encephalitis	1
Idiopathic	3
	24

TABLE III

RESULTS IN 24 CASES OPERATED ON FOR FITS

	As to Age of Patients						*As to Duration of Symptoms*					*As to Operative Procedure*			
	1-10 yrs.	*10-20 yrs.*	*20-30 yrs.*	*30-40 yrs.*	*40-50 yrs.*	*50-60 yrs.*	*1-6 mo.*	*6-12 mo.*	*1-5 yrs.*	*5-10 yrs.*	*10 plus yrs.*	*Exploration*	*Cortical Resection*	*Removal Calcified Lesion*	*Total*
No seizures			2				1				1	1	1		2
Rare seizures		2	2	1	2		1		4	1	1	3	2	1	6
Improved		3		3			1		1	2	2		6	1	7
No change	1		2	1		1	1	1	2	1		3	2		5
												7	11	2	20
P. O. death				1							1		1		1
Unable to follow or operation too recent			1	2					3			1	2		3
TOTAL	1	5	7	8	2	1	4	1	10	4	5	8	14	2	24

severity. Five cases are not now significantly better although temporary improvement or freedom from attacks were seen after operation.

It is apparent from the tables that in this small series, neither the age of the patient nor the duration of seizures before operation had any significant effect upon the results obtained. However, when the results are analyzed with regard to the procedure used (Table III), it is seen that cortical resection gave either marked or moderate improvement in eight of eleven cases as compared with similar results in fifteen of twenty cases in the entire series. Perhaps more significantly we can say that almost half (three to seven) of the cases subjected to exploration only, showed poor results as contrasted to twenty-five per cent of poor results in the entire series.

It can be concluded that epilepsy is not a surgical disease but that seizures are often a symptom of surgical disease of the brain in the form of an expanding lesion. Seizures due to localized atrophic lesions can, in properly selected cases, be stopped or greatly improved by a properly conceived and executed surgical procedure. All patients with seizures should have a thorough examination for neurologic disease. This should include not only the usual clinical examinations but also special laboratory procedures to the extent of their availability.

References

1 Brodsky, I.: "The Trephiners of Blanche Bay, New Britain, Their Instruments and Methods," *Brit. Jour. Surg.*, 26: 1-19, 1938.

2 Dudley, B. S.: Quoted by Cutter. (See seq.)

3 Brainard, D.: "Trephining for Epilepsy and Insanity," *Chi. Med. Jour.*, 16 (N. S. 2): 639-641, 1859.

4 Billings: Quoted by Cutter. (See seq.)

5 Penfield, W.: *Epilepsy and Cerebral Localization*, Charles Thomas, Springfield, 1941, 623 pp.

6 Gibbs, F. A., and Gibbs, E. L.: *Atlas of Electroencephalography*, Lew. A. Cummings Co., 1941, 221 pp.

7 Jasper, H., and Kershman, J.: "Electroencephalographic Classification of the Epilepsies," *Arch. Neurol. & Psychiat.*, 45: 903-944, 1941.

8 Parker, H. L.: "Epileptiform Convulsions: The Incidence of Attacks in Cases of Intracranial Tumor," *Arch. Neurol. & Psychiat.*, 23: 1032-1041, 1930.

9 Groff, R. A.: "The Meningioma as a Cause of Epilepsy," *Ann. Surg.*, 101: 167-175, 1935.

10 Davis, L., and Droegemueller, W.: "The Surgical Treatment of Epileptiform Seizures," *Ann. Surg.*, 103: 669-682, 1936.

11 Kocher: Quoted by Penfield. (Supra.)

12 Sachs, E.: "The Subpial Resection of the Cortex in the Treatment of Jacksonian Epilepsy (Horsley operation) with Observations on Areas Four and Six," *Brain*, 58: 492-503, 1936.

13 Furlow, L. T.: "Subpial Resection of the Cortex for Focal Epilepsy," *Jour. Am. Med. Assoc.*, 111: 2092-2095, 1938.

14 Rowe, S. N., and Watts, J. W.: "Traumatic Epilepsy with Cortical Scar: Diagnosis and Results of Operation in Twenty Cases," *Med. Annals of Dist. of Columbia*, 5: 298-301, 1936.

15 German, W. J.: "Epileptogenic Cortical Scars (Results of Surgical Removal)," *Arch. Neurol. & Psychiat.*, 41: 73-82, 1939.

16 Scarff, J. E.: "A Specific Epileptic Syndrome Relieved by Lysis of Pacchionian Granulations," *Arch. Neurol. & Psychiat.*, 36: 373-374, 1936.

17 Van Wagenen, W. P., and Herren, R. Y.: "Surgical Division of Commisural Pathways in the Corpus Callosum: Relation to Spread of An Epileptic Attack," *Arch. Neurol. & Psychiat.*, 44: 740-759, 1940.

18 Cutter, J. S.: "Benjamin W. Dudley and the Surgical Relief of Traumatic Epilepsy," *Int. Absts. Surg.*, 50: 189-194, 1930.

19 Gibbs, F. A., Lennox, W. G., and Gibbs, E. L.: "The Cortical Frequency Spectrum in Epilepsy," *Arch. Neurol. & Psychiat.*, 45: 613-621, 1941.

20 Hudson: Quoted by Brodsky. (Supra.)

21 Penfield, W.; Erickson, T., and Tarlov, I.: "Relation of Intracranial Tumors and Symptomatic Epilepsy," *Arch. Neurol. & Psychiat.*, 44: 300-315, 1940.

22 Penfield, W., and Humphrey, S.: "Epileptogenic Lesions of the Brain: A Histologic Study," *Arch. Neurol. & Psychiat.*, 43: 240-261, 1940.

23 Powers, H., and Lahey, F. H.: "The First Case in Which Abdominal Surgery Was Suggested for the Relief of Epilepsy," *Boston Med. & Surg. Jour.*, 175: 124-126, 1916.

24 Reed, C. A. L.: "Some Typical Recoveries in Iowa from Chronic Convulsive Toxemia (Epilepsy) Following Surgical Correction of the Abdominal Viscera," *Jour. Iowa State Med. Soc.*, 10: 344-345, 1920.

25 Reed, C. A. L.: "Epilepsy a Symptom of Splanchnoptosis," *Jour. Iowa State Med. Soc.*, 11: 344-345, 1921.

The Use of Intravenous Barbiturates in Determining the Prognosis in Metrazol Therapy*

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Ever since the advent of shock therapy in the treatment of various psychotic states, there has been an effort to find methods for determining prognosis. This paper is a report on the use of barbiturates for this purpose.

Lundy and Tovell[1] introduced the sulphur derivative of pentobarbital (nembutal) in 1934. This substance is marketed under the trade name of pentothal sodium. Gruhzit[2] and associates synthethized the sulphur analogue of sodium amytal (sodium isoamyl ethyl thiobarbiturate) and under the name sodium thioethamyl this preparation was used intravenously by Cullen and Rovenstein,[3] clinically. In this study only these ultra-short acting barbiturates have been used as it is desired to induce a short period of sleep and to estimate the patient's reaction both before falling asleep and on awakening. If the short actors, amytal and pentobarbital (nembutal) are used, the period of sleep is prolonged beyond an efficient period. In regard to the intravenous administration of any of the barbiturates, if skill is used and indications carefully evaluated, this route of administration may actually be considered safer than the oral, rectal or intramuscular route. In a study of this sort it is the only route which will produce the effect desired. The variability of reaction to all of the barbiturates is extreme and even more variation among psychiatric patients may be expected. With intravenous administration it is possible to abandon all attempts at preconceived dosage and give the amount necessary to produce the effect desired. With the thiobarbiturates, that is, sodium thioethamyl and pentothal, the rate of induction is so rapid that the maximal extent of depression is obtained immediately after the injection. The drugs are detoxified so rapidly a toxic dose is quickly eliminated if respirations can be maintained.

The drug should be given by the intermittent method rather than by continuous injection, thus allowing the effects of each dose to be observed before the next is given. This is an important point in our research because a marked change may be seen in the patient's psychotic symptoms between one cc. and another. A better estimation of the depth of psychosis can be made if the patient is observed intermittently. As the full effect of any one amount of drug may not appear for thirty seconds, the solution should be prepared in such strength that the injection can be given at intervals of one to one and one-half minutes. Barbiturates can be used in either five per cent or ten per cent solution; however, the five per cent solution in our hands has been found to be the most satisfactory for the so-called narco-analysis. The total dose administered is determined solely by the reaction of the patient and never by any preconceived plan. If any of the barbiturates are injected too rapidly there may be severe respiratory depression and if injected too slowly, especially with the thiobarbiturates, large amounts may fail to produce any degree of narcosis. Any previous medication such as morphine, bromide or other barbiturates particularly may markedly reduce the dosage.

It has long been known that following the administration of an anesthetic to a psychotic patient there would occur a brief period during which the patient was lucid and free of his psychotic symptoms. The ameliorating effect is not at all specific as it may occur following inhalation of carbon dioxide, the use of sodium amytal, etc. A similar response may be obtained with pentobarbital (nembutal), evipal, pentothal or sodium thioethamyl intravenously, and it is during this period that an estimate of the depth of the psychosis and the efficacy of shock therapy can be made. The patient becomes accessible and the precipitating factors or conflicts can be brought out more clearly. Results may be obtained in schizophrenia, excitement states, depressions and in fearful patients regardless of the illness. Ordinarily, there is little or no clearing with the organic brain syndromes and toxic states. The patient merely becomes more confused and in these cases a narco-

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analysis may sometimes offer material of diagnostic significance. The term narco-analysis is not new. Horsley[4] introduced this term for the use of a sedative which produces a diminution of the conscious state and a release of the inhibitory processes in order to determine the conflicts of the patient and allow psychotherapy to be given with the patient in an increased state of receptiveness. There are many discussions of this in the literature and many of the barbiturates have been recommended. In our experience, we have felt that the use of an intravenous barbiturate was effective only in those cases in which the inhibitory processes were more or less involuntary. The technique is as follows:

The patient is put to sleep slowly and his response regarding conflicts is watched during the prenarcosis stage. The patient is then allowed to sleep from three to seven minutes and is awakened and again questioned. With an interesting patient the use of the ultra-short actors has many technical advantages in that the patient can be put to sleep, awakened and put to sleep again as the situation requires. Because of the very brief duration of action of the ultra-short actors, it is feasible to vary the state of consciousness as needed during the questioning. This degree of flexibility could never be obtained with pentobarbital and amytal. Failures are usually accountable for by a too rapid injection of the drug. Because relatively small amounts of the ultra-short drugs produced marked changes in the state of consciousness, careless and inattentive injections often fail to produce the desired level of consciousness and may be dangerous. A group of forty patients was studied by means of ultra-short acting barbiturates in the methods above described and a record of the amount of clearing obtained was made.

In all cases the estimation of the prognosis was made on the basis of the response to the barbiturate that was administered rather than on the clinical symptoms which the patient presented at the time of the administration. There were a good many patients who showed a poor prognosis clinically, but their response to the drug indicated that their prognosis was good. On the other hand, in some cases where the clinical estimation showed good prognosis the drug failed to show any clearing. For example, Patient, M. H., a seventeen year old schizophrenic who was autistic and mute, cleared completely, discussed her problems freely and showed remarkable insight. Her prognosis with barbiturate was considered good. Due to her autism and mutism it would have been difficult to estimate the prognosis clinically except that her symptoms were of less than six months duration. In contrast, Patient M. A., a sixteen

DISTRIBUTION OF PATIENTS BY DIAGNOSIS

Diagnosis	*Number*	*Sub-Total*
Schizophrenia		
Paranoid	6	
Catatonic	7	
Hebephrenic	8	
Undetermined	3	
Paranoid State	2	
		26
Manic-Depressive		
Manic	4	
Hypomanic	1	
Circular	1	
		6
Psychoneurosis		
Reactive Depression	2	
Anxiety	1	
		3
Psychosis with Mental Deficiency	1	1
Psychopathic Personality	1	1
Involutional Melancholia	3	3
Total		40

DISTRIBUTION OF PATIENTS BY DURATION OF ILLNESS

Duration Years	*Number*
0-½	9
½- 1	9
2- 3	4
4- 5	7
6- 7	2
8- 9	3
10-11	2
12-higher	4
Total	40

Median length of duration 3.0 years

Age of the total group ranged from 15 to 59. The Median Age was 33.

year old schizophrenic who was autistic but excited, did not clear even under the deepest narcosis. At the point of falling asleep she developed transitory laryngospasm. On awakening she was not clear and the prognosis was considered poor. Her illness was only of one year's duration. Both these patients were treated with metrazol and the prognosis according to the barbiturate estimation was found to be accurate.

The patients whose prognosis is considered good using barbiturates are those who clear completely and are able to discuss their problems with insight. It has been observed that psychoneurotics are able to do this under barbiturates; therefore, the psychoneurotics or those with a psychoneurotic reaction in our group would tend to have a good prognosis by this method; however, metrazol does not help the psychoneurotic permanently and frequently does not help the psychoneurotic at all. However, our figures have not been corrected for the three patients who were diagnosed thus. Those patients considered to have a fair prognosis are patients who clear completely and show only partial insight into their difficulties.

One outstanding case is that of P. H., an eighteen year old boy who came in an acute schizophrenic upset. He showed many neologisms, coined words and his conversation was almost entirely a word salad. On being given a small amount of barbiturate, he continued in the manner described; however, as he became more deeply narcotized, he began to produce some intelligible language. It was only when he was carried very deeply into narcosis to the point where his speech was markedly slowed that he began to clear. He was barely able to talk because of the sedative action of the barbiturate but he expressed himself adequately at this stage. The feeling was that this patient had a very deep seated psychotic change which required the maximum hypnotic effect to release the inhibitory processes in order that he might clear. At this level he showed partial insight. In view of the depth of the narcosis before even partial clearing was obtained, it was felt that the prognosis was only fair and that it would take a considerable number of treatments to obtain any results whatsoever. This was borne out in the treatment schedule, the patient showing little improvement until the fourth or fifth shock treatment. The patient never made a complete recovery but was paroled home under supervision.

The prognosis was considered poor where the patients showing some clearing without any insight or showed some clearing with a persistence of delusions or showed more confusion. There is one other factor which changes the end results of shock therapy in relation to the barbiturate prognosis. In all those patients in the unimproved group who showed a good response to barbiturate, the immediate result of shock therapy following the cessation of treatment was good but these patients relapsed to their former psychotic state within a fairly short time. It has been thought for some time that shock therapy alone is not always sufficient to stabilize an individual who has suffered a psychotic break and the relapse of the five patients who showed a good response to barbiturate may be due to the fact that the conflict situation remained unchanged for them. However, this does not change the partial accuracy with which the prognosis can be determined with a narcoanalysis. In our series of patients we were able to obtain an accuracy of sixty-four per cent by this method.

DISTRIBUTION OF PATIENTS SHOWING IMPROVEMENT BY DIAGNOSTIC CLASSIFICATION

Diagnosis	*Improved*		*Unimproved*		*Total*	
	No.	*Pct.*	*No.*	*Pct.*	*No.*	*Pct.*
Schizophrenia	10	40	16	60	26	65
Manic-Depressive	5	83	1	17	6	15
Psychoneurosis	0	0	3	100	3	7
Psychosis with Mental Deficiency	1	100	0	0	1	3
Psychopathic Personality	0	0	1	100	1	3
Involutional Melancholia	1	33	2	67	3	7
Totals	17	43	23	57	40	100

In regard to the patient's ability to respond to the barbiturate, the diagnosis has no particular significance. It is noteworthy that the schizophrenic shows the most spectacular results as far as absolute change in responsiveness is concerned, but this is only because

DISTRIBUTION OF PATIENTS CONTRASTING RESULTS OF SHOCK THERAPY AND BARBITURATE STUDIES

	Improved				Unimproved				Total		
	No.		Pct.		No.		Pct.		No.	Pct.	
Treated	18		45		22		55		40	100	
Clear	9)	14	50)	78%	7)	8	31)	36%	16	40	
Partially Clear	5)		28)		1)		5)		6	15	55%
Unclear	4		22		14		64		18	45	

COMPARISON OF CLINICAL AND BARBITURATE SELECTION

Selection Method	Improved	Unimproved
Clinical	45%	55%
Barbiturate	64%	36%
Gain in Accuracy	19%	

clearing in a schizophrenic is a spectacular phenomena.

Summary

Ultra-short acting barbiturates, chiefly sodium pentothal and sodium thioethamyl were used intravenously in a five per cent solution by intermittent method of injection in an effort to determine the amount of clearing which could be gained in psychotic patients. In those patients where the psychosis cleared and insight was found to be present, the patients were considered to have a good prognosis with shock therapy. If the patient cleared and showed only partial insight, their prognosis was considered to be fair. Even though there was clearing present, if there was no insight or if there was no clearing, the prognosis was considered to be poor.

A correlation of the findings with the absolute results of metrazol shock therapy shows that there is a definite prognostic trend when barbiturates are used.

References

1 Lundy, S. J., and Tovell: "The Clinical Use of Pentothal," *Northwest Med.*, 33: 308, 1934.

2 Gruhzit, G. M.; Dox, A. W.; Rowe, L. W., and Dodd, M. C.: "A Pharmacological Study of Certain Thiobarbiturates," *J. Pharm. & Exper. Therap.*, 60: 125-142, (Jan.) 1937.

3 Cullen, S. C., and Rovenstein, E. A.: "Sodium Thioethamyl Anesthesia: Preliminary Report of Observations During Its Clinical Use," *Anesthes. & Analg.*, 17: 201-205, (July and Aug.) 1938.

4 Horsley, J. Stephen: "Narco-analysis," *Lancet*, 1: 55 (Jan. 4) 1936. Dorset Mental Hosp., England.

Emotional Factors in Structural Organic Change

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The ever-expanding horizon of psychiatry, the inevitable and progressive intrusion of psychological concepts in the consideration of morbid entities previously regarded as physiochemical changes and the reluctance of those who by habit of thought and training view all morbid phenomena as dependent upon physics and chemistry to tolerate or seriously consider other agencies than those demonstrable by the microscope or laboratory, leads us to call attention to the earlier views and practices and the cyclic changes in medical thought throughout the centuries of recorded medical progress. The student of medical history must be convinced that the treatment of mental disorders 4000 B. C. was on the whole just as humane and probably as effective as treatment applied at the beginning of the 20th century. The discussions of Greek philosophers reflect evidences of a fair understanding of mental states.

Hippocrates, 400 B. C., observed: "Man should know that from nothing else but the brain come joy, despondency and lamentation, and by the same organ we become mad and delirious; fears and terrors asail us, some by night and some by day." Aesculapius, 124 B. C., prescribed diet, massage, bathing, music

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and diversion, the very methods that we are employing today. It was only during the so-called modern period extending from 125 A. D. to the present time that we note the progressive evolution in the care of the mental case. We look back upon the days when mental sufferers were subjected to such therapeutic measures as bleeding, purgation, surprise baths, whirling machines, starvation, vile odors, and other cruel and apparently senseless procedures that received the endorsement of contemporary minds. We are not so remotely separated from the firm belief in witchcraft, the evil eye, the devil's claw and the Salem atrocities fully sanctioned and supported by medical opinion that we may consider our present day philosophies as unassailable or even as more accurate than those expressed and carried out by our forefathers. Frederick the Great observed: "The greatest and noblest pleasure in life is the discovery of new truths, the next is to shake off old prejudices." Each generation embraces and defends the empiricisms of its contemporaries and finds it difficult to abandon the well trodden pathway of established orthodoxy. Psychiatry as well as general medicine can claim no exception. We read with amused interest of the early treatment of diseases by amulets, charms and incantations. We are amused by the ritualistic futility of the medicine man of the aborigines, and while we scoff we must admit that these procedures that have survived through the centuries must have possessed some degree of merit or otherwise they would have passed into utter oblivion. Yet we find that they survive today in the form of faith cures represented by various healing cults which give undisputed testimony to the value of psychotherapy.

The humoral theory of Rokitanski was dissipated during the era of anatomical study and research. Virchow's dogmatic postulations as these relate to cellular pathology and the revolutionary effect of the laboratory era alike tended to crystallize current thought and sharply focus attention upon those elements and factors observable through the lens or test tube, a purely physiochemical orientation that because of its objectivity has successfully maintained and jealously defended its premises.

Alkan in 1930 postulated that psychogenic disturbances acting directly on the autonomic centers may, by disturbance of function, ultimately produce demonstrable organic change. A morphologic mechanism represented the final stage of a precedent series of causal functional changes. He asserted that intrapsychic conflicts produce spasms of smooth muscle fibers which secondarily lead to anemia and irreversible alterations in structure that constitute organic disease. The effect of continued emotional stresses in the initiation of such conditions as cardiac irritability, gastric hypermotility, gastric and duodenal ulcer, mucus colitis, spastic bowel, bronchial asthma, diabetes, thyrotoxic states, eczema, hypertension and other conditions in which recognized psychologic components must be considered, leads to the conclusion that psychologic dynamics are of equal importance with the biologic factors that have received such minute and careful study. Signal advances in our knowledge of the physiology of the hypothalamus and its control of emotional, thermal and pressor states offer a possible explanation of how emotional or psychic stimuli may eventually lead to structural organic changes.

The recognized sequence of organic structural change following continued psychogenic dysfunction is no longer doubted. This somewhat tardy recognition no doubt accounts for the added importance psychiatry has assumed in recent years in the curricula of our more advanced medical colleges.

Diseases of unknown origin are becoming less frequent in our modern lexicon as their possible or probable psychogenic causation is recognized. The not infrequent failure of somatic medicine to relieve the patient's distress by the correction or removal of determined organic pathology further sustains the contention that the successful practice of medicine must consider and treat not a specific organ or tissue alone but the whole individual in his inter-personal relationships and environmental reactions.

With progressively increasing momentum the conviction that the treatment of morbid processes is not the treatment of structure or organs alone, but of the emotional milieu that is their constant accompaniment as well, is gaining recognition. In effect this would remove psychiatry, as it is now generally interpreted, from the field of special practice. It becomes as much a part of general medical

practice as any of the basic sciences. The internist or specialist must be capable of dealing adequately with those emotional and personality problems of his patients that are alike important from a diagnostic and prognostic standpoint.

Rennie listed the most common of these conditions encountered in general practice as follows: "Vomiting, hyperthyroid like states with or without elevated B. M. R., urgency and frequency without adequate findings, hypertension relating to emotional states, weakness, fatigue, vertigo, gastro intestinal or cardiovascular complaints without adequate findings, refusal to eat or adhere to a diet, insomnia, states of anxiety, insecurity, suspicion or panic occurring in the hospital setting, overconcern over an illness with need for the patient to make adequate adjustment due to a disabling condition, refusal to cooperate, unresponsiveness, dullness or stupor, mental states associated with Addisons disease, pellagra, tuberculosis, etc."

In discussing the importance of mental and emotional factors concomitant with, or presenting manifestations simulating organic disease in which psychotherapy finds a useful place one must observe a rational conservatism. For instance the sufferer from paresis who may present what medical psychologists would term maladjustments and environmental conflicts with delusions and hallucinations, recovers under proper medical treatment and becomes fully adjusted without psychotherapy. Here physical therapy adequately meets the situation despite the fact that the manifest symptoms are psychologic. From time to time we have enthusiastically pointed out to our friends operating in the broader fields of medicine that the individual is greater than the sum of all his parts; that there is something beyond the objectivity of organs and tissues that demands consideration.

The necessity of fusion of psychiatry and somatic medicine becomes progressively more apparent as we note the unmistakable changes in the vegetative nervous system accompanied by endocrine imbalance, in many cases presenting schizophrenic reactions. The evidence adduced from the successful treatment of involutional depression by the administration of the hormones or estrogens demonstrates beyond question or doubt, the need for combined treatment and diagnosis.

Too much time has been spent in quibbling over mental mechanisms and hair-splitting classifications. These have been soothing to our ego but devoid of appreciable comfort to the patient. In the meantime the mental sufferer goes his unhappy way, a pariah. He is denied hospital privileges except in the private sanatorium or in the state hospital which is accepted only as a last resort and often when the malady has progressed to a point incompatible with a hopeful prognosis.

In the interval, general hospitals (so called) complacently continue to refuse to make provision for or to admit cases presenting mental symptoms thereby making contribution to the 100,000 who are segregated yearly in our publicly endowed institutions.

Steadily progressing research in the physiology of the endocrines in their relationship to the autonomic system will ultimately penetrate the veil of obscurity that conceals many of the fundamentals upon which the physiology of the brain cells and their proper functioning is dependent. More of proven fact and less of involved philosophical theory will provide a more authoritative background upon which the correlation of emotional components and physical change may be based.

More intensive studies in the physiology of the brain cells are at present being undertaken that will no doubt harmonize the divergent postulations that at present tend to confuse the student and will tend to further emphasize the importance of emotional factors in relation to physical morbidities.

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE 93719

Presentation

A negro aged 45 was admitted to the neurologic service of the Cincinnati General Hospital on May 1. He had been found unconscious on the floor of his room that morning. His employer, at whose home he resided, said that he had appeared to be normal on the previous morning. He had had occasional bouts of epistaxis for about three years. Sometimes this bleeding lasted as long as 12 to 24 hours. The history was otherwise uninformative, except that he had drunk alcoholic liquors to excess once or twice a week for many years. On May 2, a friend of the patient appeared at the hospital and stated that on the morning of April 30 he had seen the patient fall backward, striking his head with violence on a cement pavement. This blow rendered him unconscious for several minutes. He then arose and fell again. When assisted to his feet he was unable to stand, and was carried to his room and put to bed.

Examination — The abnormal findings on admission to the hospital were: coma, from which he could not be aroused; occasional yawning; abrasions over the left malar region and left tibia; some dried blood in the nares, and a blood pressure of 162 systolic and 102 diastolic. Divergent squint was noted; the corneal reflexes were not obtained, and the patient had left hemiparesis, which included the left lower portion of the face. The extremities were normally resistant to passive movements. The tendon reflexes were more active on the right than on the left, and the plantar responses were of extensor type bilaterally.

Roentgen examination of the skull revealed no fracture. A lumbar puncture was performed on admission to the ward. The initial pressure was 120 mm. of water; the cerebrospinal fluid was bloody, and after centrifugation the supernatant fluid was slightly xanthochromic. The fluid contained 75 mg. of protein per 100 ccm., and the colloidal gold curve was 0012321000. The Wassermann reactions of the blood and cerebrospinal fluid were negative.

Course—The patient recovered consciousness on May 2. His temperature, which was normal on admission, rose to 101.5° F. on May 2 and fluctuated between 98.6° and 102° F. until his death on May 13. The pulse rate fluctuated between 80 and 134, but was usually around 110. Twelve hours before death the pulse rate dropped rather abruptly from 115 to 60. The rate of respiration was 14 on admission; it rose to 25 shortly thereafter and gradually increased during his stay in the hospital to a high of 50 per minute, twelve hours before death.

On May 2 the patient roused from coma, and ptosis of the right eyelid was observed. At this time the right eyeball was deviated toward the right. The left hemiparesis was confirmed. The CSF pressure on this day was 80 mm. of water; only enough fluid was removed to determine that it was bloody (12,000 red cells per cmm.). After centrifugation, the supernatant fluid was xanthochromic. The CSF pressure on May 3 was 100 mm. of water. The xanthochromic fluid contained 2060 red blood cells per cmm. The colloidal gold curve was normal. On this day constant twitching of the left foot and leg was first noted (epilepsia partialis continua). These convulsions continued unabated until the operation on May 7; they did not spread to the upper extremity. The clonic movements ceased when he sank into stupor and returned when he was awakened. On May 6 the patient was stuporous and uncommunicative. The optic discs were normal. Ocular movements were possible in all directions. The left pupil was 5 and the right 4 mm. in diameter; they reacted promptly, but through a small range, to flashlight. The neck resisted flexion. Paralysis of the left extremities had become complete. The lower portion of the left side of the face was weak. Resistance of all extremities to passive movement was normal. The patient

apparently appreciated painful stimuli on both sides of the face and in all extremities. The tendon reflexes were equal in the two upper extremities; in the lower, the right knee jerk was the only reflex obtained and was hypoactive. The plantar responses were extensor on both sides, being more marked on the left than on the right. Hyperpiesis was maintained throughout the course in the hospital (the systolic pressure being between 145 and 240 and the diastolic between 95 and 140 mm. of mercury).

Operation—On May 7 four exploratory trephine openings were made in the skull. These were placed in both lateral postero-frontal and in both lateral parieto-occipital regions. On opening the dura over the right frontal lobe, a thin subdural blood clot was observed and evacuated. Nothing abnormal was seen in the other areas. The intracranial pressure was not increased.

Postoperative Course—The condition of the patient was not altered by the operation. On May 9 ptosis of the right eyelid was again obvious when the patient was aroused. The right eyeball was deviated far to the right, and rested in the outer canthus. On May 10 the patient lapsed into coma from which he could not be aroused. His pupils were equal and reacted well to flashlight. There developed signs of bronchopneumonia, with rising temperature and pulse and respiratory rates, and he died on May 13.

Differential Diagnosis

DR. MILTON ROSENBAUM: This is a simple case and yet, like all simple things, it is complex. The differential diagnosis is quite limited and one that has to be made frequently in a hospital of this sort.

I think we may start with the history, stressing its extreme importance, no matter how vague it may be. A good many physicians are apt to believe that neurologic diagnosis can be made on the clinical examination and laboratory data alone. In this case it would be impossible to make the diagnosis without the history. I feel so strongly about this point that I am willing to base my diagnosis in this case on the history alone.

The patient was 45 years old and he had drunk alcohol to excess for years, which would probably class him among the chronic alcoholic addicts. Apparently he had been in good health. On April 20 he was up and about, and that night he was seen to fall and strike his head. Why did he fall? Did he fall because of an intracerebral hemorrhage, or did he fall for some other reason, striking his head and inducing the coma of brain trauma? What are the points in favor of an intracerebral hemorrhage, and on the other hand, of brain trauma including subdural or extradural hematoma?

Against the diagnosis of intracerebral hemorrhage is the fact that the loss of consciousness was for a few minutes only. The patient then got up, although he had to be escorted to his room. When someone suffers an intracerebral hemorrhage, usually he doesn't get up again and get home so promptly. This patient regained consciousness quickly which means that he had lost consciousness by virtue of cerebral concussion or cerebral trauma. There is little question that this man regained consciousness. If he had fallen and not regained consciousness, it is likely that he would have been brought to the hospital immediately. Therefore, he had probably fallen on previous occasions and had been helped home by his friends; evidently they thought nothing of leaving him. From the history alone one is able to state rather definitely that this man lost consciousness because of a head trauma or brain concussion, and the effects were not severe enough to keep him in coma. Against the diagnosis of severe brain laceration is the fact that he recovered consciousness instead of expiring quickly without regaining consciousness. I feel that we have ruled out the diagnosis of intracerebral hemorrhage, and that severe brain laceration was unlikely.

What of the findings on the admission examination? He was in coma, which doesn't help us in the differential diagnosis. I think something might be said about the occasional yawning. In my experience yawning is not seen in overwhelming states such as intracerebral hemorrhage with rupture into the ventricles. The patient had abrasions on the face and legs; there was dried blood in the nose which probably might have been due to hypertensive nose bleeds. There was divergent squint. I would not place emphasis on the divergent squint in a patient in coma. The tendon reflexes were not obtained in the legs, possibly a sign of deepening coma, although where alcoholism is concerned, peripheral

neuritis must be considered. There was left hemiparesis and the extremities were normally resistive to passive movement.

The presence of bilateral Babinski responses is important. Where a stroke has occurred, bilateral extensor responses usually indicate the presence of hemorrhage rather than thrombosis and is of ominous portent. As far as head injuries are concerned, the presence of bilateral extensor responses signifies severe brain injury, but in this case there was another reason for this sign which will be discussed later.

This man had no roentgen evidence of fracture. Would the absence of skull fracture rule out brain injury? Of course not. Some physicians think that a person with a head injury and bloody CSF must have a fracture of the skull. Our patient had no fracture. Bloody CSF either comes from the brain itself or vessels about the brain, and bone rarely is of great moment in its production. The CSF pressure was not increased. In subdural hematoma or extradural hematoma, the pressure is usually but not always elevated. Dehydration possibly played a role in this instance. The Wassermann was negative, syphilis is not considered likely.

Let us consider the course of the illness. The patient regained consciousness the day after admission, then gradually drowsed into coma again. In patients with intracerebral hemorrhage with gross blood in the CSF, the hemorrhage usually has ruptured into the ventricles and most of these patients die rather promptly. The person with cerebral hemorrhage large enough to produce grossly bloody CSF is not likely to arouse from coma, then go into coma again. We have added to the evidence against the diagnosis of intracerebral hemorrhage; the history is incompatible, and the variance in the state of consciousness is unlike the course of a large cerebral hemorrhage.

Perhaps this man had a lacerated brain. If this patient died because of lacerated brain, he should have remained in coma and not alternated in and out of it. Some patients with lacerated brains may be wild, almost manical, and have to be restrained.

The patient had weakness of the left extremities and developed a right third nerve palsy. Did he have a Weber's syndrome? I doubt it very much. Possibly the reason for this eye sign was swelling or displacement of the uncus of the right temporal lobe and compression of the third nerve on the same side. Displacement of the brain stem with compression of the left peduncle against the tentorium might explain the bilateral pyramidal tract signs. If there was a subdural hemorrhage on the right, giving rise to signs in the left extremities, displacement of the left peduncle against the rigid tentorium could account for the third nerve lesion and the Babinski on the ipsilateral side.

A most important point in the consideration of subdural hematoma is the usual inability to localize the hemorrhage to one or the other side, except in this case. The most important sign in the clinical course was the continuous Jacksonian convulsions in the left lower extremity, indicating a cortical lesion. Was this sign the result of cortical trauma? Or was something pressing on and irritating the leg area? I don't think it likely that the fits were on the basis of severe cortical laceration for reasons stated previously.

Obviously the patient had hypertension but this does not preclude his having brain trauma.

Apparently the Neurological Service thought this patient had a subdural hematoma, and they did the usual thing in that case. There are several approaches to subdural hemorrhage; one is to trephine both lateral frontal and parieto-occipital regions. I think the staff did the right thing; they suspected subdural hemorrhage and went after it, but they didn't find it. A thin subdural clot was found on the right side, but obviously its removal did not help and the patient became comatose and died. Does that rule out subdural hemorrhage? Usually it does in the ordinary case. There is one thing just a bit different in this case, repetitive focal signs in the form of Jacksonian fits which help in the localization of the clot.

In conclusion the patient did not have an intracerebral hemorrhage. He was suffering solely from the effects of brain trauma, and the diagnosis may be brain laceration or subdural hematoma. His course is typical of subdural hematoma, which is my diagnosis. It should be located at the vertex since lateral exploration did not reveal it, and the continuous convulsion of the left foot indicates irritation high in the motor area.

Clinical Diagnosis

Cortical laceration involving the right motor area.

Dr. Rosenbaum's Diagnosis

Subdural hematoma of the right vertex.

Anatomical Diagnosis

Large midline subdural hematoma, right, lying between falx and mesial face of compressed and edematous right cerebral hemisphere. Multiple small, secondary hemorrhages in the midbrain and pons.

Pathological Discussion

DR. I. MARK SCHEINKER: A tremendous subdural hemorrhage measuring 10 cm. in antero-posterior and 4 cm. in dorso-ventral extent was found filling a cavity formed by the displacement of the mesial face of the right cerebral hemisphere away from the falx. There was marked edema of the right hemisphere with downward displacement and compression of the lateral ventricle and displacement of the corpus callosum downward and to the left. In addition there were multiple small areas of hemorrhage in the brain stem, particularly in the basilar part of the pons and the nervous tissue subjacent to the third and fourth nuclei. These hemorrhages explain the eye signs as well as the bilaterality of the pyramidal tract signs. Doctor Evans and I recently described brain stem hemorrhages in seven cases of subdural hematoma. We attempted to blame them on the compression of smaller branches of the posterior cerebral artery; a compression resulting from edema of the temporal lobe and herniation of the uncus over the edge of the tentorium.

It is certainly true that in the great majority of cases trauma is the cause of subdural hematoma. The condition occurs rather frequently in persons addicted to alcohol. This case is of special interest because of the extremely unusual localization of the hemorrhage. As you know, the typical localization of subdural hemorrhage is over the dorso-lateral surface of the hemisphere. The hemorrhage is usually enclosed in a membrane which is attached to the under surface of the dura. It is generally accepted that the bridging veins which extend from the surface of the cortex to the dura are responsible for the bleeding. They are thin-walled and rupture easily.

One doesn't know how much the factor of cerebral atrophy contributes to the possibility of the occurrence of subdural hemorrhage. Brain atrophy may lead to the retraction of brain cortex away from the dura. Therefore, the bridging veins may be stretched, at least they are relatively unprotected and would seem to be more apt to be ruptured. Another point of interest is the progression of the symptoms of subdural hematoma. Cushing and Putnam long ago suggested that this phenomenon might be due to the new formation of capillaries on the under surface of the dura, their rupture adding to the previous bleeding. A later explanation offered by Gardner is at present the accepted one. He thought that as the blood clot disintegrated the osmotic pressure increased within the subdural collection and fluid was attracted across the arachnoid membrane, increasing the subdural content. Such a process might favor the rupture of additional bridging veins since more tension would be placed on them by the additional displacement of the cerebral hemisphere.

DR. JOSEPH P. EVANS: I would like to say a few words about some complications that may occur occasionally in association with subdural hematoma. There may be associated edema of the underlying hemisphere, and the mesial portion of the swollen temporal lobe may shift over the free edge of the tentorium. The resulting tension on the third nerve may result in dilatation of the ipsilateral pupil. Obviously one may also explain the third nerve palsy on brain stem hemorrhage, as occurred in this case. In addition, however, there may occur infarction of that portion of the brain supplied by the posterior cerebral artery on the same side. The point is that such infarction causes edema, that in turn causes further shift of the brain stem to the opposite side. Sometimes the pupil dilates on the opposite side or bilateral pyramidal tract signs appear.

We should have attached more significance to the focal nature of the epilepsy and under those circumstances looked about the falx. Had we put an opening in the vicinity of the midline it is entirely probable that we would have again missed the clot.

DR. CHARLES D. ARING: Air might have been helpful.

DR. EVANS: These people are quite sick,

and one hates to do too much. Ventriculography definitely would have given us a clue.

DR. ARING: Initially we were aware that this man had a subdural hematoma. In 1938 when we saw this patient we were prone to think that the possibility of subdural hematoma had been exhausted when four exploratory burr holes were placed. Then we considered that a traumatic intracerebral hemorrhage was located in the foot area, resulting in an irritating focus.

Mental Hygiene*

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A certain amateur psychiatrist has remarked many times during the course of the present war, and especially when the outlook was so distressing for our side, that "a lot of us will probably be daffy from worry and anxiety before this thing is over with and peace returns to the world."

Fortunately, there is little to support this doleful prediction. While the strain has been, and will continue to be heavy and our nerves are kept raw by the potential tragedy that lies in the approach of the mailman or the ringing of the telephone, those who know the state of the American mind and are acquainted with the effects of the war in other countries that have known it intimately since 1939, have a reassuring message for us all. They remind us that the human mind, like the human body, is pretty tough to start with and becomes tougher when the going becomes harder. Although it would be incorrect to say that the present conflict will not have its mental casualties, as well as its physical casualties, there is believed to be little danger of a sharp increase in mental illness as a result of war worry, bombing (from which the American civilians have been mercifully spared thus far), and the fear which even the bravest experience in those anxious, silent moments before the zero hour. When the war is over, our people as a whole, civilians and fighters alike, are expected to return to the pursuits of peace with minds as sound and emotions under as good control as they were when the madmen of Europe began their work of death and devastation.

"Curiously enough," declared a spokesman for the U. S. Public Health Service, "the stress and strain of war are lesser hazards to mental health than the common, every-day emotional and mental conflicts within us. . . . Contrary to what you'd expect, the bombings (of cities) have had very little effect upon mental health. In Great Britain, for example, extensive preparations were made in anticipation of mental casualties. But when the air raids came, the people stood up to them with amazing mental stamina. In fact, some depressing and neurotic patients were improved by activities that supplanted their concern with their personal conflicts."

Explaining this apparent paradox, the United States Public Health Service spokesman declared:

"Those personal conflicts were forgotten in the larger and more vital obligations that people undertook for their country. . . . Human beings have enormous capacity to adjust themselves to the hardships of war. I think one of the things that help them is a feeling of unity they gain by working together for a common cause. This feeling of unity is a great morale builder. A joy is so much richer and a sorrow so much easier to bear when it is shared with others. On the other hand, people often find it more difficult to adjust themselves to their peacetime environment. They lack the feeling of unity with their country, or with their community, or sometimes even with their own families. Often a person lives in what we might call an isolationism of self. He narrows all his interests down to his own personal problems. Now this concentration on himself is particularly harmful to mental health."

Almost any specialist in mental and nervous conditions is able to bear witness to this

*Delivered over Radio Station WFSA, Montgomery, Alabama, June 15, 1943.
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spokesman's observations. That is especially true of those whose patients are of the wealthy classes. Women with servants to do the hard work which most other women have to do find themselves with more time on their hands than they have any use for. They become bored. With nothing to keep their minds occupied, they develop any number of purely imaginary illnesses and worry themselves almost to distraction over them. In the same way, they magnify the shortcomings of their families and friends, develop psychoses, and in general make life miserable for others. Maladjusted and spoiled, they visit psychiatrists' offices and expect them to find a cure for their condition in return for the generous fees which they are able to pay.

Although emotional upsets are by no means unknown to persons in less favored positions financially, those persons enjoy the same sort of protection against them which the spokesman for the U. S. Public Health Service mentioned as a by-product of war. They may envy their wealthier friends their money and leisure, but they find in time that they are at least partly compensated for their loss. With a house to keep in order, children to look after and keep out of mischief, family budgets to keep balanced, shopping to do and innumerable other duties to keep her mind occupied, the average middle-class woman lives a full and busy life. She does not have time for purely imaginary illnesses of either the body or the mind. And she is not likely to exaggerate any real ones which she may have. What is true of mentally and physically idle women is equally true of mentally and physically idle men, although the average man is better prepared to combat boredom than the average woman.

But psychiatrists are by no means the only members of the medical fraternity who know how greatly idleness affects the mind and emotions. Physicians on the staffs of tuberculosis sanatoria and other hospitals specializing in the treatment of chronic diseases spend much time trying to keep their patients in the proper emotional balance. Months and perhaps even years of invalidism bring problems of social adjustment which would never exist if the invalids had something to do. But they don't have anything to do, and their active minds and emotions become even more active. They become pitifully homesick. They worry because they do not get well as fast as they think they should. Trivial aches and pains which the busy person accepts as a matter of course are magnified to the proportions of major illnesses. The neighbor in the next bed, who would be an entirely acceptable companion and friend if both were not bored by idleness, is constantly saying and doing irritating things. In brief, having nothing to do gives the mind of the sick person free rein. Then it gets him into trouble.

What has been said of the idle rich and the chronically sick is equally true of any number of others—the aged, who surrender the responsibilities of middle age for the supposed comfort of the fireplace and the easy chair; the recipients of retirement incomes, who often find that happiness does not come with the shedding of one's burdens; the unemployed, who find in idleness no compensation for the loss of needed income; and many others.

From what has just been said, it may be inferred that the best way to keep up an even keel mentally and emotionally is to keep the mind busy. Like the body, it should have a certain amount of work to do in order to develop naturally. This may mean taking an interest in one's family, one's friends or one's community. It may, and probably does, mean concentrating upon one's job and doing one's best with it. There is a great satisfaction about the accomplishment of a worthwhile task, whether it be important or humble, and this satisfaction brings added confidence. Confidence, in turn, brings a healthy, cooperation, the calm facing of life's problems and a quiet determination to take them in one's stride and dispose of them—all these things make for good mental health.

One of the most promising aspects of the present-day approach to a healthy state of mind is the dissipation of much of the superstition regarding the cause and cure of mental disorders. The attitude of futility which dominated the thinking of scientists and laymen alike for many centuries is still found among certain elements of our population, but in the main it has given way to the same sort of conservative optimism which now characterizes our attitude toward cancer, tuberculosis and many other illnesses of the body which were formerly regarded as beyond

hope of recovery. We now know that diseases of the mind are due to definite causes and can be largely prevented and cured by treating those causes. Just as we combat physical illnesses by draining swamps, administering vaccine, swatting flies, and employing other well established procedure, so we combat mental sickness by resorting to measures that tend to produce mental health. Fatalism is as much out of place in the treatment of one as of the other.

Another encouraging aspect of the present outlook for mental hygiene, in wartime and in the happier days of peace, is the changed attitude on the part of the general public toward the mentally sick. Unfortunately, the old-time conception of this condition as something to be ashamed of and as source of embarrassment to one's family and friends has not been entirely dissipated. You still hear the mentally sick referred to by the unfeeling and inconsiderate term "crazy." You still hear crude jokes about people who are not normal mentally. Occasionally you even see thoughtless boys and girls "picking on" their playmates who have difficulty learning their lessons and making unkind remarks about them in their presence. But society is gradually developing a more humane and intelligent attitude toward this group of life's unfortunates. We have come far in the direction of kindness and good common sense since the days when the people of London thought it was a good Sunday afternoon's sport to visit the insane asylums, infuriate the inmates by insulting remarks and even physical pain, and listen to their cries of rage and humiliation.

The relationship between physical health and mental health—the ancients were thinking of it when they spoke of "a sound mind in a sound body"—will undoubtedly receive more and more attention from physicians and psychiatrists in future. Specialists in the field of mental health know that bodily illnesses can bring on emotional upsets and that one's mental state can be adversely affected by fatigue or even by an inadequate diet. As the already mentioned spokesman for the U. S. Public Health Service pointed out, "an important part of mental hygiene involves living in accord with physical health rules—eating the proper foods at regular times, getting plenty of sleep, sunlight and fresh air, taking regular exercise, having some relaxation, and checking on your physical condition by visiting your doctor once a year."

You may be surprised to be told that your family physician, who probably has not had special training in diseases of the mind and emotions, is in the best position to help in the solution of mental hygiene problems. It is true, however.

The general practitioner, with the whole range of human ailments as his field, cannot be expected to be an expert in this one, which probably plays a relatively small part in his practice. But he knows the patient much better than any specialist does. He knows much more about the patient's background than the world's most famous psychiatrist could ever know. He probably carries the patient's medical history in his memory, from the childhood diseases of babyhood to the operation he had a year or two ago for appendicitis or a diseased kidney. He has the patient's complete confidence, if anybody has. The stubborn wall of reticence and embarrassment which rises between the mentally sick person and the strange specialist does not exist in the friendly, chummy, informal atmosphere of the family physician's living room or private office.

During the troubled days that lie immediately ahead and the happier days that, we fondly hope, will come with the return of peace, let us give the problem of mental hygiene the attention it deserves. We shall need clear, calm, courageous minds if we as a nation are to find satisfactory answers to the staggering questions that will face us before and after victory is achieved.

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DIRECTORY OF MEDICAL SPECIALISTS

It has been announced that, because of the war, the next edition of the "Directory of Medical Specialists" will not be published until 1945. However, a supplemental list of all those who have been certified by the American Boards since the last (1942) edition of the Directory, totaling about 3,600 will be issued.

This is to be distributed at cost, and monthly or bimonthly bulletins listing successful candidates for certification at examinations during the additional interim before the next edition, are to be issued as a subscribers' service.

Dr. Paul Titus (Pittsburgh) of the American Board of Obstetrics and Gynecology will continue as the Directing Editor, and Dr. J. Stewart Rodman (Philadelphia) of the American Board of Surgery continues as Associate Editor. The Editorial Board will be composed, as before, of the Secretaries of the fifteen American Boards.

Communications should be addressed to the Directing Editor, Directory of Medical Specialists, 919 No. Michigan Avenue, Chicago (11), Illinois.

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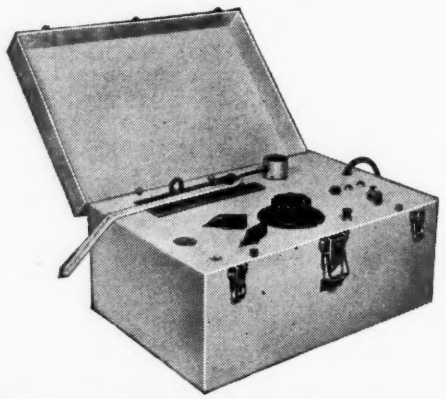

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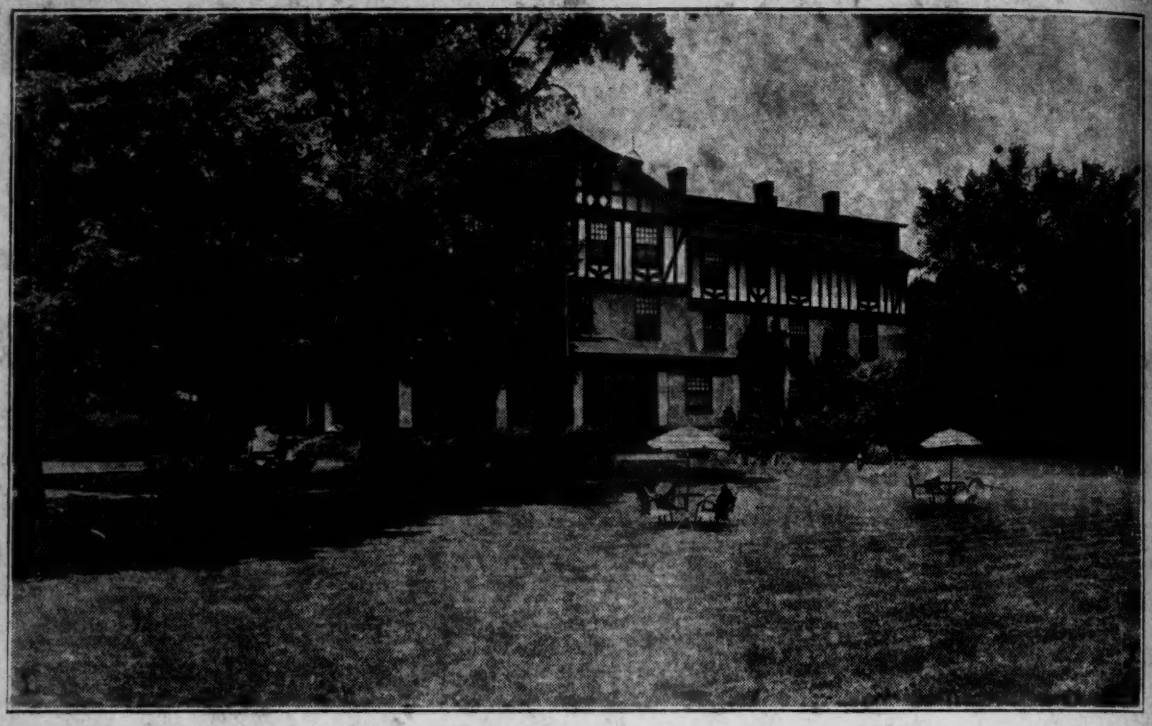

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